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De novo giant A2 aneurysm following anterior communicating artery occlusion

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Abstract

Background: *De novo* intracranial aneurysms are reported to occur with varying incidence after intracranial aneurysm treatment. They are purported to be observed, however, with increased incidence after Hunterian ligation; particularly in cases of carotid artery occlusion for giant or complex aneurysms deemed unclippable.

Case Description: We report a case of right-sided *de novo* giant A2 aneurysm 6 years after an anterior communicating artery (ACoA) aneurysm clipping. We believe this *de novo* aneurysm developed in part due to patient-specific risk factors but also a significant change in cerebral hemodynamics. The ACoA became occluded after surgery that likely altered the cerebral hemodynamics and contributed to the *de novo* aneurysm. We believe this to be the first reported case of a giant *de novo* aneurysm in this location. Following parent vessel occlusion (mostly of the carotid artery), there are no reports of any *de novo* aneurysms in the pericallosal arteries let alone a giant one. The patient had a dominant right A1 and the sudden increase in A2 blood flow likely resulted in increased wall shear stress, particularly in the medial wall of the A2 where the aneurysm occurred 2 mm distal to the A1-2 junction.

Conclusion: ACoA preservation is a key element of aneurysm surgery in this location. Suspected occlusion of this vessel may warrant closer radiographic follow-up in patients with other risk factors for aneurysm development.

Key Words: A2, aneurysm, anterior communicating, *de novo*, giant, occlusion

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INTRODUCTION

Since Graf and Hamby first coined the phrase "*de novo*" aneurysm, in 1964,[17] multiple reports have followed in an attempt to explain this phenomenon where new aneurysms arise at anatomically distinct locations that were previously normal. Different authors have presented the rate of *de novo* intracranial aneurysms (DNIA) as 0.15–4.15% per year.[8,14,25,27,35,60,63] While their pathophysiology is not completely understood, genetic, environmental, and hemodynamic risk factors are thought to contribute to their formation.[6,7,11,25,49] Changes to the cerebral hemodynamics following arterial occlusion (particularly

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of the carotid artery) have been associated with increased incidence of DNIA. A personal history of multiple intracranial aneurysms (IA) has also been found to be common in multiple series.[4,8,63]

Many postcarotid occlusion DNIAs are small when discovered and typically occur at the anterior communicating artery (ACoA), posterior communicating artery (PCoA), internal carotid artery (ICA)-bifurcation, and basilar bifurcation.[1] The post communicating segment (A2) of the anterior cerebral artery (ACA) is a very rare location for a DNIA and giant DNIAs themselves are also uncommon. There are also very few reports of giant distal ACA (DACA; post A1-2 junction) in general. We report what we believe to be the first case of a giant DNIA of the DACA. We hypothesize that a significant contributing factor to this DNIA was the iatrogenic occlusion of the ACoA during previous ACoA aneurysm clipping 6 years earlier. We will review other DNIA risk factors and patient/case specific details that may have also contributed to this unusual aneurysm.

CASE REPORT

Initial presentation and surgery

A 46-year-old female was incidentally found to have an ACoA aneurysm during screening conducted because her mother and maternal uncle both suffered subarachnoid hemorrhage (SAH). Of note, her mother's SAH was due to giant MCA aneurysm. The patient smoked tobacco daily and was normotensive. She had no other medical illnesses. A computed tomography angiogram (CTA) uncovered a 3 mm ACoA aneurysm facing posteriorly with the dome directed to the left [Figure 1]. The right A1 was dominant. The patient underwent a right lateral supraorbital craniotomy for clipping. The aneurysm was largely excluded using a single curved clip, but there was a small remnant based on the right half of the ACoA that was unable to be included in the blades. Bipolar electrocautery was used to coagulate this portion until it was occluded. The patient awoke at her neurological baseline. Postoperative CTA did not reveal any residual aneurysm or DNIA [Figure 2].

Postoperative course and discovery of *de novo* intracranial aneurysms

The patient's last CTA prior to the discovery of her DNIA was 10 months after surgery at which time there was no new or recurrent aneurysm. She lost to follow-up after this, until 2014, when she presented to the neurosurgery clinic complaining of subjective memory issues. She was neurologically intact. The patient reported that she continued smoking following the initial surgery. A new CTA revealed a 2.5 cm × 3.7 cm × 3.0 cm largely thrombosed, partially calcified DNIA based off of the medial wall of the right pericallosal artery [Figure 3].

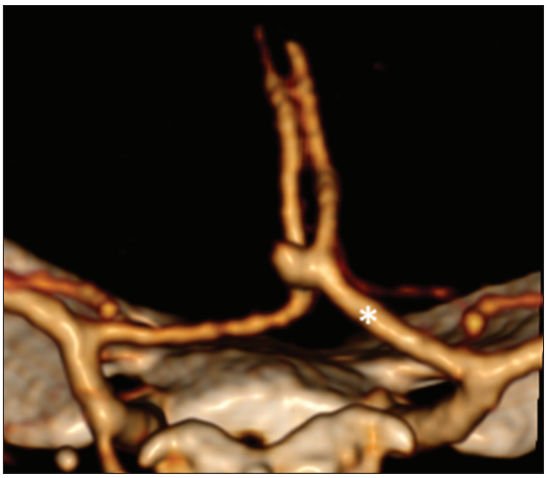

Figure 1: Computed tomography angiogram three-dimensional reconstruction of the initial anterior communicating artery aneurysm as viewed from a posterior/superior angle. The right A1 (white asterisk) is dominant. The aneurysm is saccular and projects posteriorly and to the left

A digital subtraction angiogram (DSA) was obtained to further evaluate the lesion. It revealed small mural filling of a giant aneurysm from the right A2 2 mm distal to the A2 takeoff without any ACoA filling [Figure 4]. After a discussion of all options including surgical, endovascular observation, the patient elected to proceed with the endovascular approach. A 2.5 mm × 20 mm Flex Pipeline Embolization Device (PED; [Coviden; Plymouth, Minnesota, USA]) was deployed in the proximal right A1 extending across the aneurysm neck into the distal right A2 [Figure 5]. Angiography following stent deployment revealed immediate stagnation in the filling portion of the aneurysm.

The patient was neurologically intact after the procedure. There were no complications. She currently reports that her memory issues are unchanged. She was discharged home on Aspirin and Plavix.

DISCUSSION

We present the first case of a giant DACA DNIA. This aneurysm was felt to be *de novo* as opposed to recurrent or secondary to dissection for several reasons. The neck small remnant of the ACoA aneurysms (ACoAA) was based on the ACoA itself. It was coagulated with bipolar electrocautery as originally described by Yasargil[67] and later demonstrated in other series to be effective in treating aneurysms smaller than 3 mm in size.[40] In addition, the origin of its neck was clearly distal to the previously clipped ACoAA with a neck based on the A2 as opposed to ACoA. While an iatrogenic A2 dissection at the initial surgery was considered, the lack of A2 stenosis on CTA immediately after surgery or at the 10-month follow-up made this less likely.

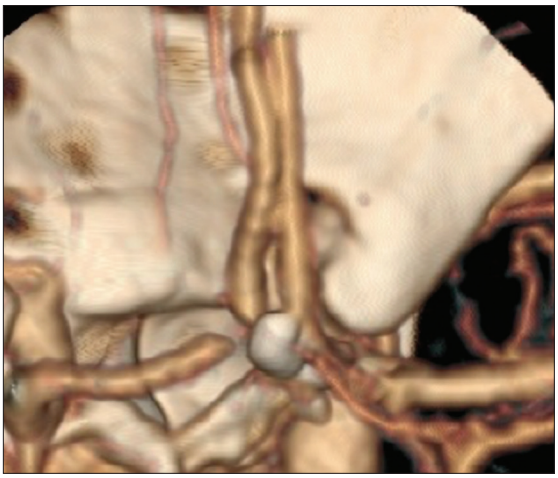

Figure 2: Computed tomography angiogram three-dimensional reconstruction of the anterior communicating artery complex postoperatively as viewed from a posterior/superior angle. The curved clip is seen occluding the aneurysm. There is no remnant or new aneurysm

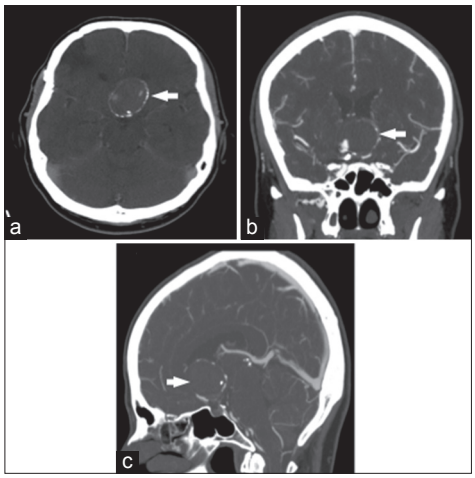

Figure 3: Computed tomography angiogram 6 years following the initial anterior communicating artery aneurysm clipping shows the 2.5 cm × 3.7 cm × 3.0 cm giant aneurysm (white arrows). There is calcification and thrombus evident on axial (a), coronal (b) and sagittal (c) cuts. There is some filling of the aneurysm from the right A2 seen on the coronal view

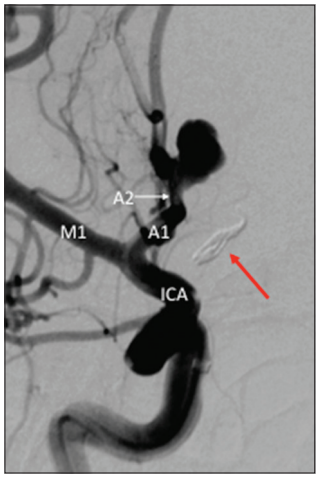

Figure 4: Digital subtraction angiogram viewed from an anterior-oblique angle. The aneurysm is based on the medial wall of the right A2 approximately 2 mm distal to the A1-A1 junction. The anterior communicating artery, patent at the initial surgery, does not fill at all now. The aneurysm clip (red arrow) is seen in the region the anterior communicating artery aneurysm and artery to be used

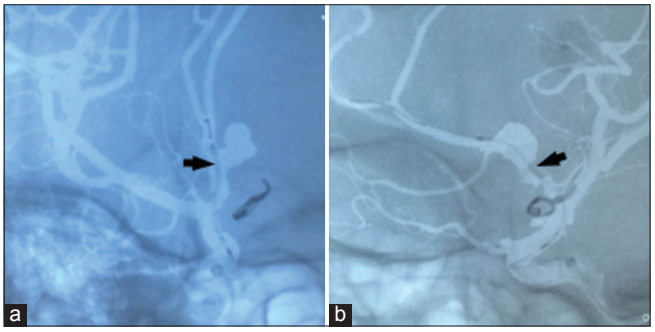

Figure 5: Digital subtraction angiogram (a and b) during Pipeline Embolization Device deployment. The microcatheter can be seen traversing the right internal carotid artery, right A1 and terminating in the distal right A2. Black arrows indicate where it crosses the aneurysm base. There was stagnation noted within the aneurysm immediately following Pipeline Embolization Device placement

All DACA aneurysms (distal to the ACoA) are rare and comprise only 2–9% of all -[9,22,23,30,37,41,46,51,54,65,68,70] A2 aneurysms represent approximately 5% of pericallosal aneurysms and 0.2–1% of all IA.[31] DACA aneurysms are typically small; Lehecka *et al.*[31] conducted an angiographic analysis of 101 patients with these aneurysms and found that unruptured aneurysms in this location are approximately 4.2 mm and ruptured ones are 7.4 mm. Giant aneurysms of the ACA distal to the ACoA are exceedingly rare with only 30 cases reported in the literature.[2,9,10,13,20,21,26,28,31,32,36,37,39,42,43-45,50,52-54,59,61,66]

The development of DNIAs is thought to be multifactorial and include many of the risk factors that lead to the initial aneurysm. Women and smokers have been shown to be at increased risk.[25,27,29,58] This particular patient was both a woman and continued to smoke following her initial aneurysm surgery despite advice to abstain. She also had a family history of SAH. Bor *et al.*[3] found that individuals with 2 first degree family members were at considerable risk for development of a first aneurysm during follow-up screening despite 2 previously negative screens and 10 years of follow-up. It is reasonable to extrapolate this data to the formation of *de novo* aneurysms; particularly in high-risk patients with a family history and other risk factors for DNIA. Other risk factors for DNIA formation include a personal history of multiple aneurysms[4,8,63] and hypertension[48] although the latter has been inconsistently supported in reports.

The interval between initial aneurysm treatment and DNIA development is also debatable. Some authors report DNIA detection over 18 years from the initial aneurysm treatment.[4,35,63,69] There are also reports of

DNIA developing and rupturing very soon after aneurysm treatment.[11,18] Wang *et al*.[62] found that 6 of 9 DNIAs were detected within the first 2 years following aneurysm surgery in patients with regular angiographic surveillance with only 1 DNIA being found after 10 years. In patients without regular surveillance, 8 of 15 DNIA were detected 7 years after the initial aneurysm surgery or later. Our patient presented 6 years after the initial ACoA aneurysm clipping. She had no surveillance imaging in the interim. The issue of surveillance imaging to monitor for DNIAs is also contested with some authors advocating for it[58] and others asserting there is insufficient evidence to do so.[62]

We felt that this patient's ACoA aneurysm was appropriately excluded and did not require any further follow-up after her 1-year appointment. She may however, represent a small subset of high-risk patients that despite a low incidence of DNIAs in most aneurysm patients could be considered for close follow-up. This patient in particular was prone to DNIA formation as she is female, a smoker, and has a family history of SAH.

The location of this DNIA is unique. Increased hemodynamic stress within the cerebral circulation, particularly following Hunterian ligation, has been reportedly associated with a significant increase in the rate of DNIA formation.[1,38,56,57,64] When compounded with the other risk factors for DNIA mentioned above, a change in cerebral blood flow patterns and increased wall stress may be a likely culprit for the increased incidence. While therapeutic carotid sacrifice for complex aneurysms of the ICA has been associated with an increased incidence of DNIA, there have been no reports of DNIA secondary to ACoA occlusion and no reported post carotid ligation DNIA of the DACA region. We initially thought that a giant aneurysm maybe arising from a recurrent ACoA aneurysm after we obtained the CTA. After reviewing the DSA, however, we discovered that the ACoA was completely occluded up to the A1-2 junction and that the base of the aneurysm was actually 2 mm distal to the A1 bifurcation on the medial wall of the right A2 [Figure 4]. This location is directly in the path of the increased blood flow that was redirected from the occluded ACoA. Animal studies have shown up to 9-fold increase in blood flow through the basilar artery after carotid ligation demonstrating that vessel occlusion causes a significant shift in hemodynamics.[16] In support of this idea, Arambepola *et al*.[1] found that the majority of reported DNIAs after carotid ligation were discovered in the contralateral carotid distribution. In this instance, the blood was rerouted through the A2 because of the location of the arterial closure. Additionally, high wall shear stress (WSS) has been implicated in aneurysm formation.[34] This ACoA occlusion coupled with a dominant right A1 likely placed an increased hemodynamic burden on the A2 thereby playing a significant role in the development of this patient's DNIA [Figure 6]. While the blood flow across the ACoA is significantly less than the carotid, the increased flow and subsequent WSS that is, redirected from a newly occluded ACoA may make the A2 susceptible to damage and *de novo* formation.

Lending further to the impact of hemodynamics is the observed angle between the A1 and A2 arteries. Ingebrigtsen *et al*.[24] studied branch angles at the MCA bifurcation, basilar bifurcation and ICA bifurcation and found that an observed angle between the parent vessel and largest branch of 61–115° was associated with increased likelihood of aneurysm discovery by 3.46 times. While the study did not have enough A1-2 junctions to be included in the final report, the senior author feels that the above statistical significance likely applies to this region as well (via personal communication). In our particular patient, the increased flow was directed through an observed angle of 64.7° [Figure 7].

The treatment of complex, giant aneurysms of the pericallosal region can be challenging. Open microsurgical techniques include direct clipping, trapping with bypass, or distal occlusion with bypass. A bypass for this type of aneurysm would likely be A3-to-A3. PEDs are typically used for complex and giant cavernous or intradural ICA aneurysms proximal to the PCoA but further indications are being explored. Puri *et al*. recently published a small experience using PEDs in distal aneurysms including 2 in the pericallosal region.[47] While an open microsurgical approach would have allowed for immediate exclusion and debulking of the aneurysm, PEDs have also been reported to decrease the size of large aneurysms.[55] Despite reports of early success, the Pipeline device and other flow-diverting stents are not without their own set

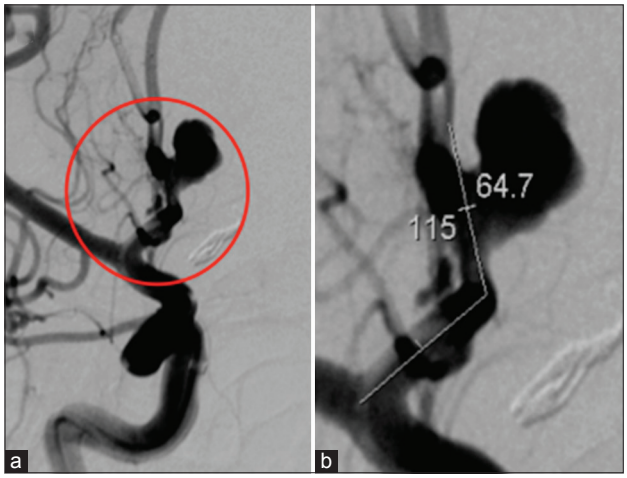

Figure 6: Digital subtraction angiogram showing the branching angle between the right A1 and A2 vessels. The anatomy within the red circle (a) is enlarged on the right (b). The observed angle as measured by Ingebrigtsen *et al*.[24] is the angle measured between the largest branching vessel (A2 in this case) and the trajectory the parent vessel would have taken had it continued. It is 64.7° here

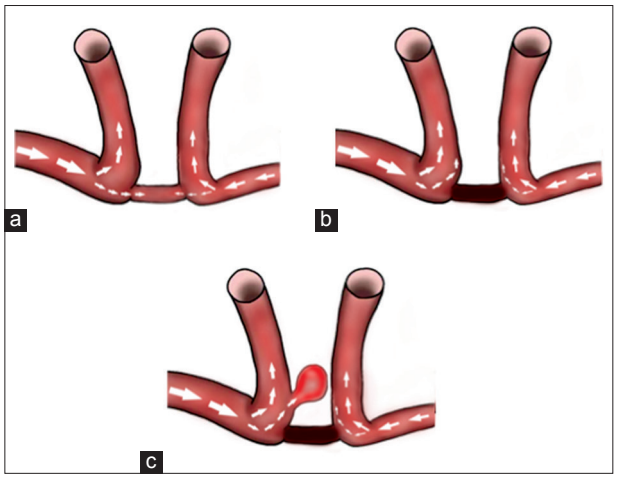

Figure 7: Characterization of blood flow patterns in the anterior cerebral artery/anterior communicating artery complex. The thickness of the arrows indicates amount of flow. A patent anterior communicating artery (a) would distribute the flow from the dominant right A1 such that some would flow into the anterior communicating artery (hence the leftward directed aneurysm) and some would flow into the A2. Once the anterior communicating artery is occluded (b), all of the flow from the A1 is directed into the A2. This eventually contributes to aneurysm formation (c)

of shortcomings and complications.[5,12,15,19,33] Both open and endovascular options should be considered in the context of each specific patient scenario. Thus, patients with complex lesions such as this one should be treated at highly specialized centers capable of offering all the potential treatment alternatives.

CONCLUSION

We report a novel case of a giant A2 DNIA. While this patient had multiple risk factors for DNIA development, the location of her *de novo* aneurysm just distal to the occluded ACoA suggests that the altered hemodynamics played a significant role in development of her giant aneurysm. PED indications are expanding but they are associated with complications and their long-term outcomes are not known. ACoA preservation is a key element of microsurgical treatment of aneurysm in this location. The ACoA likely became occluded due to a combination of manipulation, clip application, and excessive bipolar cautery. Patients in whom the ACoA is suspected of occlusion after aneurysm surgery should be considered for closer radiographic follow-up; especially if they possess other risk factors for aneurysm development.

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Conflicts of interest
There are no conflicts of interest.

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